UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
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Reply To: OEA095

MEMORANDUM **DRAFT**

SUBJECT: **Proposed Risk Assessment Approach for Evaluating Potential Risks From Consuming Breast Milk**
Portland Harbor Superfund site

FROM: Government Risk Assessment Team

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TO: Lower Willamette Group

INTRODUCTION

One of the remaining questions we have for the Portland Harbor human health risk assessment is how to evaluate potential risks to infants from consuming chemicals in breast milk as a result of maternal exposure from eating contaminated fish. EPA and its partners evaluated the feasibility of conducting a risk assessment based on exposure to breast milk using EPA's *Human Health Risk Assessment Protocol for Hazard Waste Combustion Facilities*[1] (Combustion Guidance), *Exposure Factors Handbook*[2], *Child-Specific Exposure Factors Handbook*[3], and examples from other hazardous waste sites, such as the Housatonic river project in Massachusetts[4]. We determined that it is feasible to include exposure to breast milk in the Portland Harbor Superfund human health risk assessment (PH HHRA), and that this is an important exposure pathway.

To assist the Lower Willamette Group in incorporating the breast milk consumption pathway into the human health risk assessment, we prepared this memorandum to present the relevant exposure and risk equations, and exposure and toxicity parameters (summarized in Table 1). We include example calculations using total polychlorinated biphenyl (PCB) Aroclors to show how the various equations in EPA's combustion guidance can be modified to focus on the fish consumption pathway. The actual risk assessment should include all relevant chemicals, such as PCB congeners, chlorinated dibenzo-*p*-dioxins, chlorinated dibenzofurans, and DDT and its degradation products.

Generally, risk assessments are limited to an evaluation of risk, and do not consider comparative risks or benefits. For example, eating fish is health beneficial compared with

eating other protein sources. Public health agencies commonly address the health tradeoffs of eating contaminated fish, but the issue is not typically discussed in a Superfund risk assessment. For breast feeding, however, the benefits to infants are so substantial that we consider it appropriate to discuss the issue in the risk assessment. We therefore present a section on the risks and benefits of consuming contaminated breast milk. We request that this information be included in the Portland Harbor risk assessment.

PROPOSED RISK ASSESSMENT APPROACH

Calculated Exposure to Infants

We mainly relied on the equations presented in the EPA combustion guidance document[1], modified to make the equations no longer specific to dioxins or the inhalation pathway, and instead make them appropriate for fish consumption. The key concept is that the concentration of a chemical in milk can be calculated from the long-term body burden in the mother. This is consistent with the information presented in the Agency for Toxic Substances Disease Registry (ATSDR) *Toxicological Profile for Polychlorinated Biphenyls*[5].

We start with the average daily intake of chemicals from fish consumption (modified from Table C-1-4 of the Combustion Guidance[1]):

$$ADD_{mother} = \frac{C_{fish} \times IR_{fish} \times CF \times F_{fish}}{BW_{af}}$$

Where:

ADD_{mother} = Average daily dose to mother (mg/kg/day)
C_{fish} = Chemical concentration in fish (assume 1 mg/kg for PCBs)
IR_{fish} = Ingestion rate of fish (subsistence rate of 142.4 g/day)
CF = Conversion factor (0.001 kg/g)
F_{fish} = Fraction of fish contaminated (1)
BW_{af} = Body weight (66 kg for average adult female)

The ingestion rate used in the example is that being used in the Portland Harbor HHRA for fishers subsisting on resident fish. The site risk assessment should include all of the other relevant fish consumption rates being used for the Portland Harbor HHRA. The fish consumption rate is an annualized rate (*i.e.*, it includes the assumption that fish are eaten throughout the year, so exposure frequency, exposure duration, and averaging time are not included in the equation). Loss of chemicals during cooking has been considered at other sites, but is not included in EPA's Combustion Guidance or as a part of the Portland Harbor HHRA Risk Characterization. For body weight, we consider it appropriate to use the average female weight of 66 kg, rather than the guidance value of 70 kg (average adult weight).

For this example, the calculations are performed assuming a total PCB concentration of 1 mg/kg in whole-body tissue. This value is within the range of PCB concentrations measured in Portland Harbor resident fish composites and was chosen primarily for illustrative purposes. The actual risk assessment should use chemical concentrations appropriate for the various species of fish sampled.

$$ADD_{mother} = 1 \text{ mg/kg} \times 142.4 \text{ g/day} \times 0.001 \text{ kg/g} \times 1 / 66 \text{ kg} = 0.0022 \text{ mg/kg/day}$$

EPA has found that dietary intake of PCBs during pregnancy and lactation is only weakly correlated with PCB concentrations in human milk. The more important determinant is long-term consumption. The following equation is used to calculate the PCB concentration in milk fat.

$$C_{milkfat} = \frac{ADD_{mother} \times h \times f_1}{\ln(2) \times f_2}$$

Where:

$C_{milkfat}$ = PCB concentration in milkfat (mg/kg-lipid)
ADD_{mother} = Average daily dose to mother (mg/kg/day)
h = Half-life of PCB (7 years = 2555 days)
f_1 = Fraction of ingested PCB stored in fat (0.9)
f_2 = Fraction of mother's weight that is fat (0.3 kg-lipidBW/kg-totalBW)

$$C_{milkfat} = \frac{0.0022 \text{ mg/kg-totalBW/day} \times 2555 \text{ days} \times 0.9}{0.693 \times 0.3 \text{ (kg-lipidBW/kg-totalBW)}}$$

$$= 24 \text{ mg/kg-lipid}$$

The equation was modified from Table C-3-1 of the Combustion Guidance[1], and is consistent with equations 1 through 3(b) in Section 3.4.4.2 of the ATSDR *Toxicological Profile*[5]. The equation is for steady-state conditions, and therefore we assume that maternal intake occurs over a time-period greater than the PCB half-life. We also assume that PCB concentrations in breast milk reflect the maternal body burden. For a derivation of the equation for $C_{milkfat}$, see Attachment A.

Average daily doses to the infant are calculated separately for carcinogenic and noncarcinogenic effects. For carcinogenic effects, the average daily dose is the following (modified from Table C-3-2 of the Combustion Guidance[1]):

$$ADD_{ca\text{-}infant} = \frac{C_{milkfat} \times IR_{milk} \times f_3 \times f_4 \times ED_i \times EF_i}{AT_i \times BW_i}$$

Where:

$ADD_{ca\text{-}infant}$ = Average daily dose for breast-feeding infant (mg/kg/day)
$C_{milkfat}$ = Concentration of chemical in milk fat (mg/kg-lipid)
IR_{milk} = Ingestion rate of breast milk (0.69 kg/day)
f_3 = Fraction of breast milk that is fat (0.04)

f_4 = Fraction of ingested PCB that is absorbed (0.9)
ED_i = Exposure duration of breast-feeding infant (1 year)
EF_i = Exposure frequency of breast-feeding infant (365 days/year)
AT_i = Averaging time – carcinogen (70 years x 365 days/year)
BW_i = Body weight of breast-feeding infant (9.4 kg)

$$ADD_{ca\text{-}infant} = \frac{24 \text{ mg/kg-lipid} \times 0.69 \text{ kg/day} \times 0.04 \times 0.9 \times 1 \text{ yr} \times 365 \text{ day/yr}}{70 \text{ yr} \times 365 \text{ day/yr} \times 9.4 \text{ kg}}$$

$$= 0.00091 \text{ mg/kg/day}$$

For non-cancer effects, the average daily dose is the following (modified from Table C-3-2 of the Combustion Guidance[1]):

$$ADD_{nc\text{-}infant} = \frac{C_{milkfat} \times IR_{milk} \times f_3 \times f_4 \times ED_i \times EF_i}{AT_{nc} \times BW_i}$$

Where:
$ADD_{nc\text{-}infant}$ = Average daily dose for breast-feeding infant (mg/kg/day)
$C_{milkfat}$ = Concentration of chemical in milk fat (mg/kg-lipid)
IR_{milk} = Ingestion rate of breast milk (0.69 kg-milk/day)
f_3 = Fraction of breast milk that is fat (0.04 kg-lipid/kg-milk)
f_4 = Fraction of ingested PCB that is absorbed (0.9)
ED_i = Exposure duration of breast-feeding infant (1 year)
EF_i = Exposure frequency of breast-feeding infant (365 days/year)
AT_{nc} = Averaging time – non-carcinogen (= ED_i x EF_i)
BW_i = Body weight of breast-feeding infant (9.4 kg)

$$ADD_{nc\text{-}infant} = \frac{24 \text{ mg/kg-lipid} \times 0.69 \text{ kg-milk/day} \times 0.04 \text{ kg-lipid/kg-milk} \times 0.9 \times 1 \text{ yr} \times 365 \text{ day/yr}}{1 \text{ yr} \times 365 \text{ day/yr} \times 9.4 \text{ kg}}$$

$$= 0.063 \text{ mg/kg/day}$$

ATSDR considers exposure of one year or more to be chronic exposure. However, EPA's Superfund program defines seven years or more as chronic exposure[6]. Therefore, we included an alternative child exposure with one year of breast-feeding exposure, and six years of fish consumption, for a total child exposure period of seven years.

$$ADD_{child} = \frac{\frac{C_{milkfat} \times IR_{milk} \times f_3 \times f_4 \times ED_i \times EF_i}{BW_i} + \frac{C_{fish} \times IR_{fish} \times CF \times ED_c \times EF_c}{BW_c}}{AT}$$

Where:
ADD_{child} = Average daily dose for breast-feeding and fish-eating child (mg/kg/day)
$C_{milkfat}$ = Concentration of chemical in milk fat (mg/kg-lipid)

C_{fish} = Chemical concentration in fish (assume 1 mg/kg for PCBs)
IR_{milk} = Ingestion rate of breast milk (0.69 kg-milk/day)
IR_{fish} = Ingestion rate of fish (60 g/day)
f_3 = Fraction of breast milk that is fat (0.04 kg-lipid/kg-milk)
f_4 = Fraction of ingested PCB that is absorbed (0.9)
ED_i = Exposure duration of breast-feeding infant (1 year)
EF_i = Exposure frequency of breast-feeding infant (365 days/year)
ED_c = Exposure duration of child (6 years)
EF_c = Exposure frequency child (365 days/year)
BW_i = Body weight of breast-feeding infant (9.4 kg)
BW_c = Body weight of child (15 kg)
AT = Averaging time (non-carcinogen = 7 years infant and child)
(carcinogen = 70 years)

Using this approach, the calculated child exposure to PCBs for carcinogenic risk is

$$ADD_{ca\text{-}child} = 0.0012 \text{ mg/kg/day}$$

And the calculated child exposure to PCBs for non-carcinogenic risk is

$$ADD_{nc\text{-}child} = 0.012 \text{ mg/kg/day}$$

Calculated Risk to Infants

Using the standard risk characterization equations, excess lifetime cancer risk and non-cancer hazards are calculated separately. Excess lifetime cancer risk is approximated by:

$$ELCR_{infant} = ADD_{infant} \times SF_o$$

Where:
$ELCR_{infant}$ = Excess lifetime cancer risk to infant from breast feeding
SF_o = Cancer slope factor – oral [2 $(mg/kg/day)^{-1}$ for total PCBs]

$$ELCR_{infant} = 0.00091 \text{ mg/kg/day} \times 2 (mg/kg/day)^{-1} = 2 \times 10^{-3}$$

Using the longer exposure period of seven years for a child, the calculated ELCR is essentially the same value.

$$ELCR_{child} = 0.0012 \text{ mg/kg/day} \times 2 (mg/kg/day)^{-1} = 2 \times 10^{-3}$$

Where:
$ELCR_{child}$ = Excess lifetime cancer risk to infant from breast feeding and child from eating fish

The non-cancer hazard quotient is:

$$HQ_{infant} = \frac{ADD_{infant}}{RfD}$$

Where:
HQ_{infant} = Hazard quotient for breast-feeding infant
RfD = Non-cancer reference dose (2×10^{-5} mg/kg/day for total PCBs)

$$HQ_{infant} = 0.063 \text{ mg/kg/day} / 2 \times 10^{-5} \text{ mg/kg/day} = 3{,}200$$

Using the longer exposure period of seven years for a child, the calculated hazard quotient is:

$$HQ_{child} = 0.012 \text{ mg/kg/day} / 2 \times 10^{-5} \text{ mg/kg/day} = 600$$

Another approach to evaluate less-than-chronic exposure to a child is to use ATSDR's minimal risk level (MRL, comparable to an RfD) of 3×10^{-5} mg/kg/day for intermediate-duration (subchronic) oral exposure to PCBs. ATSDR defines intermediate-duration exposure as two weeks to one year. The intermediate-duration MRL was derived using data on monkeys that approximated exposure during breastfeeding. For this reason, it may be a better indicator of toxicity than the chronic RfD (which is equal to the chronic MRL). Using the intermediate-duration MRL, the calculated hazard quotient is:

$$HQ_{infant} = 0.063 \text{ mg/kg/day} / 3 \times 10^{-5} \text{ mg/kg/day} = 2{,}100$$

Comparison of Calculated Risks with Acceptable Levels

Using the approach presented in this memorandum, the excess lifetime cancer risk is approximately 2×10^{-3} for an infant consuming total PCBs in breast milk for one year. This is above the cancer risk range of 10^{-4} to 10^{-6}, the target range within which the EPA strives to manage risks as part of a Superfund Cleanup. The acceptable excess lifetime cancer risk under Oregon Department of Environmental Quality rules[7] is 1×10^{-6}.

For non-cancer effects, the hazard quotients range from 600 to 3,200 depending on which calculation method is used. For hazard quotients above 1, unacceptable exposures may be occurring and there may be concern for potential non-cancer effects. Under Oregon rules, the acceptable hazard quotient is 1. Generally, the greater the magnitude of the hazard quotient above 1, the greater the level of concern for non-cancer health effects.

The calculated cancer risks and non-cancer hazards are based on a total PCB concentration in whole-body resident fish composites of 1 mg/kg. Although this concentration was used as a convenient value to demonstrate the calculations, it is within the range of total PCBs in resident fish composites in the initial study area of the lower Willamette River. Reasonable maximum exposure whole-body smallmouth bass concentrations of PCBs by river mile ranged from 0.25 mg/kg to 4.5 mg/kg. The site-

wide reasonable maximum exposure PCB concentration in whole-body common carp is 5.9 mg/kg, which would result in a hazard quotient of 18,000 given the same exposure assumptions used for smallmouth bass. Because the calculated excess lifetime cancer risk and hazard quotient are considerably above acceptable levels, we conclude that infant exposure to chemicals in breast milk is an important pathway for the Portland Harbor human health risk assessment.

The risk levels associated with the breast feeding pathway are similar to, or well above those associated with direct consumption of fish by adult subsistence fishers. For consumption of whole-body smallmouth bass, the calculated risks presented in Appendix F, Table 5-35 of the Portland Harbor Round 2 Report[8] range from excess lifetime cancer risks of 4×10^{-4} to 8×10^{-3}, and hazard quotients of 30 to 500 for adult fish consumption at 142 grams per day.

Uncertainty Evaluation

Following standard guidance, the risk assessment for this pathway should include an evaluation of the associated uncertainties. During our evaluation of this pathway, we considered the following.

The only exposure to infants evaluated was consumption of breast milk. We did not consider other potential exposure routes, such as transplacental transfer of PCBs from mother to fetus during pregnancy.

The PCB RfD is based on LOAELs developed from studies on monkeys. The health effects included inflammation of glands in the eye, distorted growth of finger and toe nails, and decreased antibody responses. The uncertainty factors used in the derivation of the human health RfD total 300, applied to an animal LOAEL of 0.005 mg/kg/day. The calculated HQ from consumption of breast milk is from 2 to 10 times greater than the uncertainty factor.

Another uncertainty is the application of the RfD to one year of exposure, rather than long-term (lifetime) exposure. EPA's Superfund guidance defines chronic exposure as that between seven years and a lifetime. However, in its Combustion Guidance[1], EPA considered it appropriate to apply the chronic RfD to one year of exposure to breastmilk, at least for screening purposes. Application of the chronic RfD to one year of exposure may also be appropriate considering the potential sensitivity of infants to adverse health effects. As presented above, alternative approaches to evaluating non-carcinogenic risks for exposure periods less than a lifetime could reduce the calculated hazard quotient by a factor of 5. Using the intermediate-duration MRL instead of the chronic RfD would reduce the calculated hazard quotient by a factor of 1.5.

We also looked at the reduction in body burden of PCB during a year of breast feeding to see if that could result in reduced concentrations in breast milk. If the concentration in milk fat (C_{milk} = 24 mg/kg-lipid) is equivalent to the concentration in other tissues (C_{lipid}), then the body burden in the mother is:

C_{lipid} x BW_{af} x f_2 =

24 mg/kg-lipid x 66 kg-BW x 0.3 kg-lipid/kg-BW = 480 mg PCB

The loss of mass during one year of breast feeding is:

IR_{milk} x $C_{milkfat}$ x f_3 x 365 days =

0.69 kg/day x 24 mg/kg-lipid x 0.04 kg-lipid/kg-milk x 365 days = 240 mg PCB

This implies that a mother will lose approximately half of her PCB body burden (240 mg / 480 mg) during a year of breast feeding, assuming that there is no additional consumption of contaminated fish during this period. This simplistic evaluation is consistent with EPA's determination (summarized in the GE/Housatonic report[4]) that there will be a 20 percent reduction of PCBs in the mother every three months. Over a year, this would correspond to a reduction of $1 - (1 - 0.2)^4 = 0.6$, or a 60 percent reduction in PCB mass after one year. The reduction in mass (and concentration) averaged over the course of the year would be about half of this value. If we assume that the PCB concentration in breast milk reduces to one-half the original value in one year, then the average concentration consumed by the infant over the year would be about three-quarters of the original concentration. The corresponding risk and hazard calculations would be lower by this amount. Refining the calculations to include this reduction in mass would reduce the calculated hazard quotient by a factor of about 1.3.

At other sites, including the Housatonic River site[4], EPA presented the potential risks from breast milk consumption as a ratio to background risk rather than as an excess lifetime cancer risk or hazard quotient. The background total PCB concentration used for the Housatonic River site is 0.32 mg/kg-lipid in milk. Using the assumed total PCB concentration of 1 mg/kg in Portland Harbor fish tissue and the assumed subsistence fish consumption rate, the calculated total PCB concentration in breast milk is 24 mg/kg-lipid. As an alternative presentation of risk in the uncertainty section, this result can be discussed as corresponding to a risk 75 times that of the background concentrations used for the Housatonic River site.

EPA is aware that in the lower Willamette River, consumption of resident fish by lactating mothers is already discouraged by the PCB fish advisory[9]. The Oregon Department of Human Services (DHS) advisory states that:

> Women of childbearing age, particularly pregnant or breastfeeding women, children and people with weak immune systems, thyroid or liver problems, should avoid eating resident fish from Portland Harbor, especially carp, bass and catfish.

For this reason, there may currently be limited infant exposure to breast milk contaminated as a result of consumption of resident fish in the lower Willamette River. In

addition, DHS advice on preparing fish for consumption, including removing fat from fillets (rather than consuming whole-body fish), could substantially lower risks to fish consumers, and also subsequently to breast-feeding infants. However, the results presented here appear to quantitatively support the advisory, and indicate that there are potentially unacceptable risks by the breast-feeding pathway.

HEALTH CONSULTATION ON BREAST-FEEDING PATHWAY

EPA asked the Oregon Environmental Health Assessment Program (EHAP, formerly SHINE) to develop recommendations on how to address the potential health risks for infants exposed to PCBs in breast milk in the context of the many health benefits of breast feeding. The following sections summarize the results of health consultation.

Background

Consuming resident fish species from the harbor has been declared a public health hazard, and correlated fish advisories have been issued[9]. Despite the current advisory, subsistence fishing from the harbor may occur, although the extent to which it occurs is unknown. Without considering the health benefits of breast milk, preliminary estimates suggest that PCB levels in the milk of a woman eating fish resident to Portland Harbor could pose a health risk to nursing infants.

The breast feeding exposure pathway for environmental contaminants presents unique challenges to the health/risk assessor and public health officials. In most health/risk assessments, the exposure medium is considered only a delivery vehicle for the contaminant of concern. In the case of breast milk, however, the exposure medium contains a multitude of healthful compounds that have been well documented to produce measurable health benefits. In fact, not breast feeding is considered a risk factor for several acute and chronic health conditions. Therefore, consideration of this exposure pathway requires not a simple assessment of risk, but rather, a balancing of the risks associated with contaminant exposure against the well documented health benefits of breast feeding. To further complicate this process, there is no accepted threshold value for PCBs in breast milk. In the absence of such thresholds, local, state, and federal health agencies struggle to formulate an appropriate public health response to this potential threat.

Health Benefits of Breast Feeding

Breast feeding has been shown to be the healthiest option for infants under most conditions. Breast milk is an inexpensive, ideally balanced source of nutrition[10]. The infant immune system is matured and bolstered by breast milk components. Immunoglobulin A (IgA) in breast milk reduces the uptake of dietary antigens, protecting against development of food allergies[11]. IgA in breast milk also protects the infant against microbes from the maternal gut and prevents microbes from binding to the intestinal mucosal surface[12]. Breast milk also has anti-inflammatory properties,

stimulates maturation of the intestinal epithelium and enhances the protective character of the intestinal mucosa[13]. This overall enhancement of immune function means reduced risk of multiple types of infectious disease for the infant.

Breast feeding is also associated with improved IQ scores and neurological development and reduced risk of SIDS, type I and type II diabetes, leukemia, obesity, asthma, and high cholesterol[10]. Recent research suggests that exclusive breast feeding may reduce the risk of celiac disease[14]. There are also psychological benefits to the improved mother-infant bonding that accompanies consistent breast feeding. Overall, non-breast-fed babies have a 21 percent higher mortality rate than breast-fed babies[10].

Mothers who breast feed also enjoy health benefits including reduced postpartum bleeding, reduced risk of breast and ovarian cancer, easier loss of excess adipose accumulated during pregnancy, and enhanced psychological well-being with increased bonding between mother and child. Breast feeding also benefits society by reducing health care costs (healthier babies), increasing worker productivity (children sick less often), and introduces less waste into the environment[10].

Evaluation of Contaminated Breast Milk

Despite the documented benefits of breast feeding, breast milk may also contain environmental contaminants such as PCBs. PCBs may accumulate in the adipose tissue of mothers who are exposed to them. Upon lactation, body lipids and PCBs accumulated there over the course of several years are mobilized and secreted into milk. As discussed above, if a mother were to consume 142 g/day (5 ounces/day) of resident fish from Portland Harbor containing 1 ppm PCBs, EPA calculations estimated breast milk PCB levels in excess of 24 mg/kg-lipid (generally reported as µg/g-lipid in public health literature).

An infant nursing from a mother with 24 µg/g-lipid PCBs in her milk is estimated to get 0.063 mg/kg/day. In other media, adverse health effects would be expected at this dose because it is over 10 times higher than the lowest dose (0.005 mg/kg/day) shown to cause health effects in monkeys. Health effects that occurred in monkeys at this dose include altered finger and toe nails and nail beds, inflammation of eye-lid glands, and decreased immunity[5].

The estimated 0.063 mg/kg/day PCB dose to infants is within the range of the lowest levels (0.02-0.08 mg/kg/day) that caused more serious health effects in monkeys. These included decreased platelet volume, increased eye exudate, severely altered finger and toenails, and decreased performance in spatial learning memory and discrimination problem tests[5].

The 0.063 mg/kg/day PCB dose is just below the lowest levels (0.1-0.2 mg/kg/day) shown to cause more severe health effects in monkeys. These include hair and nail loss, anemia, liver damage, swelling of the cells in the gall bladder and biliary duct, facial edema, conjunctivitis, gingival necrosis, and thyroid desquamation[5].

Comparison of Calculated Breast Milk Levels with Measured Breast Milk Levels

The example calculation of breast milk PCB concentration (24 µg/g-lipid) using a high fish consumption rate of whole-body fish exceeds documented levels measured in human breast milk. Calculating exposure using a fish ingestion rate of 17.5 g/day would result in PCB breast milk levels comparable to the 0.247 µg PCB/g-lipid background level used by ATSDR[5]. The Housatonic River study[4] used a background PCB concentration of 0.32 µg/g-lipid. Most studies found subtle health effects in children including deficits in composite activity rating with breast milk PCB concentrations greater than 0.62 µg/g-lipid and a negative correlation between breast milk PCB concentrations and performance on standardized neurocognitive tests and or altered immunological parameters[5]. Table 2 (adapted from Table A-1 in ATSDR's Toxicological Profile for PCBs)[5] summarizes some of the potential health effects associated with measured PCB concentrations in breast milk.

In most cases, toxicity was attributed to prenatal exposure to PCBs. One study, known as the "Dutch PCB/Dioxin Study,"[15,16] compared the neurological performance of children exposed to PCBs only prenatally with that of children exposed prenatally and postnatally via breast milk. While children consuming milk containing higher PCBs fared worse than children consuming milk with lower levels, all groups of breast-fed children fared better than bottle-fed children. The lowest performing children had been exposed to high levels of PCBs prenatally but had been formula fed after birth. This seems to suggest that breast feeding, even with PCB-contaminated milk, served to counter the negative effects of prenatal PCB exposure[15]. The studies cited in this report conclude that, even at the highest breast milk PCB levels measured, the health benefits of breast feeding still outweigh the risks associated with contaminant exposure.

Example calculated breast milk PCB concentrations related to Portland Harbor (24 µg/g-lipid), however, exceed moderate background levels (0.247 µg/g-lipid). The highest PCB concentrations measured in breast milk that EHAP was able to find in the literature was 15 µg/g-lipid[17]. While this study by Hara, et. al.[17] identified more health effects in children who breast-fed for more than 5 months from mothers with extensive occupational PCB exposure histories, these effects were self-reported, and none of the children were diagnosed as having PCB poisoning by health care professionals. Additionally, health effects in children were correlated to occupational exposure history for their mothers as opposed to measured breast-milk PCB levels.

Risk vs. Benefit

If a PCB dose of 0.063 mg/kg/day were estimated in any other media, EHAP would recommend that citizens reduce or eliminate their exposure to that medium. However, PCB exposure via breast milk necessarily follows additional prenatal exposures during critical developmental windows. Studies cited here suggest that breast milk, even with significant PCB contamination, may serve to reverse or stabilize developmental lesions initiated by prenatal exposure[5].

The primary goal for environmental and health agencies should be to reduce PCB exposure to women of childbearing age. These findings reinforce the importance of current fish advisories issued by Oregon's Office of Environmental Public Health (OEPH)[9]. However, the recommended course for infants who have already had prenatal exposure to PCBs is clear. Breast feeding is best for infants regardless of PCB levels in the milk.

Affected Population and EHAP Activities

In regards to the Portland Harbor Superfund site, the affected population (subsistence fish eaters who are pregnant, planning on becoming pregnant, or nursing) includes hard-to-reach ethnic communities. Since 2002, EHAP has worked with community-based organizations and local agencies to identify affected populations and provide information to them about safe fish consumption. EHAP encountered several barriers in this effort. The primary barrier was a lack of resources to locate and build relationships with high fish consumers. Other barriers included communicating information in the appropriate language. While the current findings reinforce the importance of conducting this kind of outreach, EHAP does not currently have the resources to continue these time-intensive efforts.

Public Health Conclusions

- For lipophilic environmental contaminants such as PCBs, the nursing infant receives the highest dose of contaminant and is the population most sensitive to that contaminant.

- Breast milk containing PCB concentrations equal to or greater than 24 µg/g-lipid is as much as 75 times higher than background levels in the general population. However, due to the significant benefits of breast milk, breast feeding should still be recommended.

- Elevated levels of PCBs in breast milk indicate significant prenatal exposure to PCBs.

- The current fish advisory is protective of nursing infants as long as their mothers adhere to it.

- Because remediation will not likely reduce PCB levels below health-based guidelines for several decades, effective risk mitigation depends on adherence to current fish advisories. Lack of resources for community outreach and education regarding fish advisories limits the effectiveness of those advisories to protect public health.

Public Health Recommendations

- The Lower Willamette Group (LWG) and EPA should include the breast milk exposure pathway in the baseline human health risk assessment using methodology presented above.

- EPA and LWG should include language in the baseline human health risk assessment encouraging women to continue breast feeding regardless of contaminant exposure unless directed otherwise by their physician. This language should include information on the well-documented health benefits of breast feeding.

- ~~If funding is available, the Oregon Office of Environmental Public Health (OEPH) should sample the breast milk of women with an environmental history that includes exposure to PCBs. This biomonitoring data should be made available to health care providers to guide them in early intervention efforts to treat potential health conditions initiated by the prenatal PCB exposures indicated by elevated breast milk levels.~~

- The Lower Willamette Group (LWG) should fund the Office of Environmental Public Health (OEPH) to conduct a sustained community outreach campaign directed towards women of childbearing age who are high fish consumers. This campaign should promote breast feeding as the healthiest option for infants regardless of the mother's exposure scenario, promote fish as a healthy source of nutrition, but discourage eating resident fish species from Portland Harbor such as bass, carp, and catfish. To effectively encourage these health-protective behaviors, the outreach campaign should:

    - Identify affected populations (i.e., ethnic or cultural groups that report frequent consumption of locally caught fish)
    - Characterize affected populations as to:
        - Effective communication channels
        - Beliefs, attitudes, and knowledge about breast feeding and environmental contaminants in the fish they consume
        - Fishing practices (species and parts of fish consumed, locations fished, frequency, preparation methods)
    - Develop culturally appropriate strategies and messages to encourage desired behaviors in target populations
    - Implement the strategies and disseminate the messages that have been developed in the manner determined to be most effective for target populations
    - Evaluate effectiveness of the campaign by assessing behavior changes in target populations

ENDNOTES

[1] U. S. EPA. *Human Health Risk Assessment Protocol for Hazard Waste Combustion Facilities.* (EPA 530-R-05-006, September 2005.
[2] U.S. EPA. *Exposure Factors Handbook.* National Center for Environmental Assessment, Office of Research and Development. August 1997.
[3] U.S. EPA. *Child-Specific Exposure Factors Handbook.* National Center for Environmental Assessment, Office of Research and Development. EPA-600-P-00-002B, Interim Report. September 2002.
[4] U.S. Army Corps of Engineers, U.S. EPA. *Human Health Risk Assessment, GE/Housatonic River Site, Rest of River,* Volume 1. February 2005.
[5] Agency for Toxic Substances and Disease Registry (ATSDR). *Toxicological Profile for Polychlorinated Biphenyls* (Update, November 2000).
[6] U.S. EPA. *Risk Assessment Guidance for Superfund.* Volume 1. Human Health Evaluation Manual (Part A). Interim Final. (EPA 540-1-89-002). December 1989.
[7] Oregon Administrative Rules, Chapter 340, Division 122, Section 0115.
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Table 1
Parameters for Evaluation of Risk from Consuming Breast Milk

Parameter	Units	Description	Value[a]
ADD_{mother}	mg/kg/day	Average daily dose to mother	Calculated
$ADD_{ca\text{-}child}$	mg/kg/day	Average daily dose to child (cancer)	Calculated
$ADD_{nc\text{-}child}$	mg/kg/day	Average daily dose to child (non-cancer)	Calculated
C_{fish}	mg/kg	Chemical concentration in fish	Calculated from site data. Assume 1 for example.
IR_{fish}	g/day	Ingestion rate of fish	142.4 for subsistence fishers[b]
IR_{milk}	kg/day	Ingestion rate of breast milk	0.69
CF	kg/g	Conversion factor	0.001
F_{fish}	unitless	Fraction of fish contaminated	1
BW_{af}	kg	Body weight of adult female	66[c]
BW_i	Kg	Body weight of infant	9.4
$C_{milkfat}$	mg/kg-lipid	Concentration in milkfat	Calculated
h	days	Half-life of chemical	2555 (7 years) for PCBs
f_1	unitless	Fraction of ingested chemical stored in fat	0.9 for PCBs
f_2	unitless	Fraction of mother's weight that is fat	0.3
f_3	unitless	Fraction of breast milk that is fat	0.04
f_4	unitless	Fraction of ingested chemicals that is absorbed	0.9 for PCBs
ED_c	year	Exposure duration of breast-feeding child	1
EF_c	days/year	Exposure frequency of breast-feeding child	365 days/year
AT_c	days	Averaging time – carcinogen	25550 (70 years)[d]
AT_{nc}	days	Averaging time – non-carcinogen	= ED x EF
$ELCR_{child}$	risk	Excess lifetime cancer risk	Calculated
HQ_{child}	hazard	Hazard quotient	Calculated
SF_o	$(mg/kg/day)^{-1}$	Cancer slope factor – oral	2 for PCBs
RfD	(mg/kg/day)	Reference dose	2×10^{-5} for PCBs
MRL	(mg/kg/day)	Minimal risk level (intermediate duration)	3×10^{-5} for PCBs

Notes:

a) Exposure assumptions taken from *Human Health Risk Assessment Protocol for Hazard Waste Combustion Facilities* (EPA 530-R-05-006, September 2005), except as noted.
b) One of the higher ingestion rates used in the Portland Harbor risk assessment.
c) EPA combustion facilities guidance uses 70 kg (average weight of male and female adults).
d) EPA combustion facilities guidance is to use 1 year. We considered this too conservative, and used the lifetime AT_c value typically used at Superfund sites.

Table 2
Health Effects in Human Infants Associated with PCBs in Breast Milk[a]

Study	Mean Breast Milk PCB Conc. (µg/g-lipid)	ADD $infant_{nc}$[b] (mg/kg/day)	Observed Health Effects[c]	Comparison with Formula-fed Controls
Michigan Cohort	0.87 (fisheaters) 0.62 (nonfisheaters) Total PCBs	0.0023 (fisheaters) 0.0016 (nonfisheaters)	Reduced birth weight, head circumference, and gestational age in newborns. Neurobehavioral alterations in newborn and older children.	Deficits correlated with prenatal exposure but not postnatal exposure via breast milk.
Dutch Cohort	0.62 Total PCBs	0.0016	Reduced birth weight. Reduced growth during first 3 months in formula-fed, but not breast-fed children. Neurobehavioral alterations and changes in T-lymphocyte subpopulations and thyroid hormone levels in infants.	Slight increased incidence of mild hypotonia and neurological function in children breastfed with high PCBs relative to formula fed, but mental performance was enhanced with breastfeeding regardless of PCB contamination. Minor effects associated with postnatal exposure via breast milk resolved by 18 months of age.
German Cohort	0.43 Sum of PCB congeners[d]	0.0011	Neurodevelopmental and thyroid hormone alterations in infants.	Breast-fed children did better than formula-fed in all parameters tested.
Inuit Infant Study	0.62 Sum of PCB congeners[d]	0.0016	Immunologic alterations.	No difference in immunological parameters between breast fed and formula fed infant
North Carolina Cohort	1.8 Sum of PCB congeners[d]	0.0048	Neurobehavioral alterations in infants	No comparison.
Intermediate-duration MRL[e] for Aroclor 1254:		**0.00003 mg/kg/day**		

Notes:

a) Based on Table A-1 from ATSDR Toxicological Profile for PCBs[5].

b) Non-cancer Average Daily Dose to infant via breast milk. Parameter not reported in studies, but doses were calculated for infants nursing from mothers with mean breast milk PCB concentrations reported. This exposure pathway is not applicable to formula-fed infants. (See Appendix A for calculations and assumptions). It is important to note that any exposure via breast milk follows an unquantified prenatal exposure.

c) No distinction between effects due to prenatal exposure and effects due to postnatal exposure via breast milk (unless otherwise noted in table).

d) PCB value is the sum of three non-dioxin-like congeners (PCB 138, PCB 153, and PCB 180).

e) MRL = minimal risk level for intermediate-duration exposure (two weeks to one year).

Attachment A

Derivation of Equation for Chemical Concentrations in Milkfat

The EPA combustion facility guidance document[1] and ATSDR's Toxicological Profile[2] do not elaborate on the derivation of the equation for calculation of chemicals present in milkfat. The main EPA reference for the equation is from an evaluation of infant exposure to chlorinated dibenzodioxins and chlorinated dibenzofurans in breast milk[3]. In this attachment, we explicitly derive the steady-state equation used to approximate chemical concentrations in maternal body fat, which is assumed to be equivalent to the concentration in breast milk.

The chemical body burden in the mother is calculated assuming first-order kinetics:

$$B_t = B_0 \, e^{-kt}$$

Where:
t = Time period (years)
B_t = Body burden at time t (mg)
B_0 = Body burden at time $t = 0$ (mg)
k = Rate constant = $\ln(2) / h$ (days^{-1})
h = Half life of chemical in body (days)

Using a similar approach, the maternal daily chemical intake, m (mg/kg/day), is used to calculate the concentration of chemical in the mother's tissue. The contribution to maternal chemical levels (C_{mother} in mg/kg-body-weight) is:

$$C_{mother} = \int_0^T me^{-kt} \, dt$$

where the mother is exposed to chemicals in fish from time $t = 0$ to time $t = T$ (in days). The general solution to this equation is:

$$\int_0^T me^{-kt} \, dt = \frac{me^{-kT}}{-k} - \frac{me^0}{-k} = \frac{me^{-[\ln(2)/h]T}}{-k} - \frac{m}{-k} = \frac{me^{-[\ln(2)][T/h]}}{-k} + \frac{m}{k}$$

$$= \frac{m\,(0.5)^{T/h}}{-k} + \frac{m}{k} = \frac{m}{k}\left[1 - (0.5)^{T/h}\right]$$

[1] U. S. EPA. *Human Health Risk Assessment Protocol for Hazard Waste Combustion Facilities.* EPA 530-R-05-006, September 2005.
[2] ATSDR. *Toxicological Profile for Polychlorinated Biphenyls.* November 2000.
[3] Allan H. Smith. Infant Exposure Assessment for Breast Milk Dioxins and Furans Derived from Waste Incineration Emissions. *Risk Analysis,* Vol. 7, No. 3. 1987.

Substituting again for $k = \ln(2) / h$,

$$C_{mother} = \frac{mh}{\ln(2)}[1 - (0.5)^{T/h}]$$

If the exposure period of the mother to contaminated fish (T) is equal to the chemical half-life (h) of 7 years for PCBs, then the chemical concentration in the mother's tissue is:

$$C_{mother} = 0.5\frac{mh}{\ln(2)}$$

If the exposure period of the mother to contaminated fish is equal to four half-lives ($T = 4h = 28$ years), then the chemical concentration in the mother's tissue is:

$$C_{mother} = 0.94\frac{mh}{\ln(2)}$$

The limit of $[1 - (0.5)^{T/h}]$ for large values of T (relative to the half-life h) is 1. Therefore, at exposure periods to the mother longer than the chemical half-life, a reasonably conservative assumption is that the chemical concentration in the mother can be approximated by:

$$C_{mother} = \frac{mh}{\ln(2)}$$

This equation is further refined by considering the fraction of the chemical stored in fat tissue (f_1) and the fraction of the mother's weight that is fat (f_2).

$$C_{mother} = \frac{mh}{\ln(2)}\frac{f_1}{f_2}$$

Substituting the symbol ADD_{mother} for m, and assuming that the chemical concentration in milkfat is equivalent to the chemical concentration in the mother's lipid tissue, yields the equation for $C_{milkfat}$ shown in the main text.

$$C_{milkfat} = \frac{ADD_{mother}\ h}{\ln(2)}\frac{f_1}{f_2}$$